Repair of Dissecting Aneurysm of the Ascending Aorta

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In the case presented, a prior diagnosis of "heart failure" was corrected by cardiac catheterization and other studies revealing the aortic aneurysm. The surgical correction is described and illustrated, and the various types and characteristics of dissecting aneurysms of the aorta are discussed. Underscored is the belief that diagnosis should be followed as rapidly as possible by definitive surgery.

Case Presentation

In June 1973 a 41-year-old white man was admitted to Methodist Hospital for evaluation and treatment. Six months earlier the patient, a marine engineer who was then on a voyage, had suddenly developed a severe, crushing chest pain that radiated into his jaws and teeth. He became short of breath and was placed in bed where he remained ill and unable to eat for the four days it took his ship to make port in rough weather. During this time his chest pain subsided somewhat, but he remained dyspneic.

Hospitalized as soon as he debarked, he came to feel reasonably well during a two-week stay. Soon after leaving the hospital he went back to work but could not continue since he again experienced shortness of breath and also swelling of the legs and abdomen. A physician told him at this time that he "was in heart failure" and prescribed the usual drugs. Although the patient showed some improvement on this regime he remained unable to work. He was then referred to a large metropolitan hospital for more definitive diagnosis and treatment. It was now some five months since the initial episode.

A series of studies, including cardiac catheterization, demonstrated a dissecting aneurysm of the ascending aorta. Although the point of origin of the aneurysm could not be determined precisely, it appeared to extend proximally to involve the aortic valve, which showed signs of gross incompetence. Distally, the aneurysm appeared to extend up to the innominate vessels without involving them. The chest roentgenogram showed the broadening and thickening of the aorta typical of this type of aneurysm.

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In the interval between these studies and his admission to Methodist Hospital, Houston, the patient remained at bed rest on a regimen to control heart failure. There was no recurrence of chest pain and the patient generally felt better but it was apparent he would not be able to return to work. Any attempt at physical exertion brought a prompt recurrence of shortness of breath.

Our evaluation of the patient confirmed the other hospital's diagnosis of a dissecting aneurysm of the ascending aorta. It was clear from arteriograms made available to us and a number of general features, moreover, that the aneurysm was what we have termed a type II dissecting aneurysm, that is, one limited to the ascending aorta and stopping short of the innominate vessels. An important nonspecific feature pointing to this diagnosis was the patient's marfanoid appearance: He was 6'3" in height and had always been considered tall for his age. In addition, he had correspondingly long bones of the arms, legs, feet, and hands and hyperextensibility of the joints, all common features of Marfan's syndrome. Thus, even though all the stigmata of Marfan's syndrome were not found, it seemed probable that the patient also had degenerative disease of the arterial media (or cystic medial necrosis of the aorta), which is characteristic of the syndrome, and that the dissecting aneurysm was secondary to this condition. The fact that the patient was a male was relevant also, since, in our experience with dissecting aneurysm, men outnumber women by about five to one.

Our evaluation also confirmed the presence of aortic incompetence, an almost invariable concomitant of this type of aneurysm, with severe biventricular failure, most probably due to the left ventricular failure. There was generalized cardiac enlargement on chest x-ray, with more marked enlargement of the left ventricle associated with vascular congestion. In addition, these studies showed some pleural effusion bilaterally, with interseptal edema at the sides of both lungs.

The results of other studies contributed few positive findings to the diagnosis, but were consistent with it and tended to verify the nature and extent of the aneurysm. The patient gave no history of hypertension, a frequent but not invariable concomitant of a dissecting aneurysm. There were no irregularities in cardiac rhythm, no brachial blood pressure differential, no C N S symptoms, all of these findings being consistent with an aneurysm limited to the ascending aorta below the innominate vessels. Peripheral pulses were normal and no bruits were heard. There was slight ankle edema, as might be expected in aortic insufficiency with heart failure, and bilateral varicose veins.

On auscultation there was a loud S3 gallop heard best at the apex and a short systolic ejection murmur that was loudest in the aortic area and did not radiate to the neck, findings consistent with involvement of the ascending aorta and aortic anulus. In addition, there was a grade III to IV early diastolic murmur of semilunar insufficiency, probably of the Austin-Flint type. E C G studies revealed a single premature contraction, judged a probable fusion beat, as well as left axis deviation of the Q R S and nonspecific S T/T wave changes. In addition, there was poor R wave progression, suggestive of an anterior infarction.

In short, the fact that the patient's difficulties had started in an almost classical fashion for the condition, with a clear-cut episode of severe, tearing chest pain, together with the fact that he had been in obvious heart failure ever since, plus the positive as well as nonspecific findings that we have outlined, placed the diagnosis beyond dispute. The patient thus faced a choice between continuing with medical measures, which could at best palliate his heart failure while confronting him with the ever-present possibility of hemorrhagic catastrophe, or definitive surgery to repair the aneurysm and replace the incompetent aortic valve. Such surgery, it was pointed out, would in all likelihood enable him to return to a normal life and gainful employment. He chose the latter. Both procedures were carried out as described in the following pages.
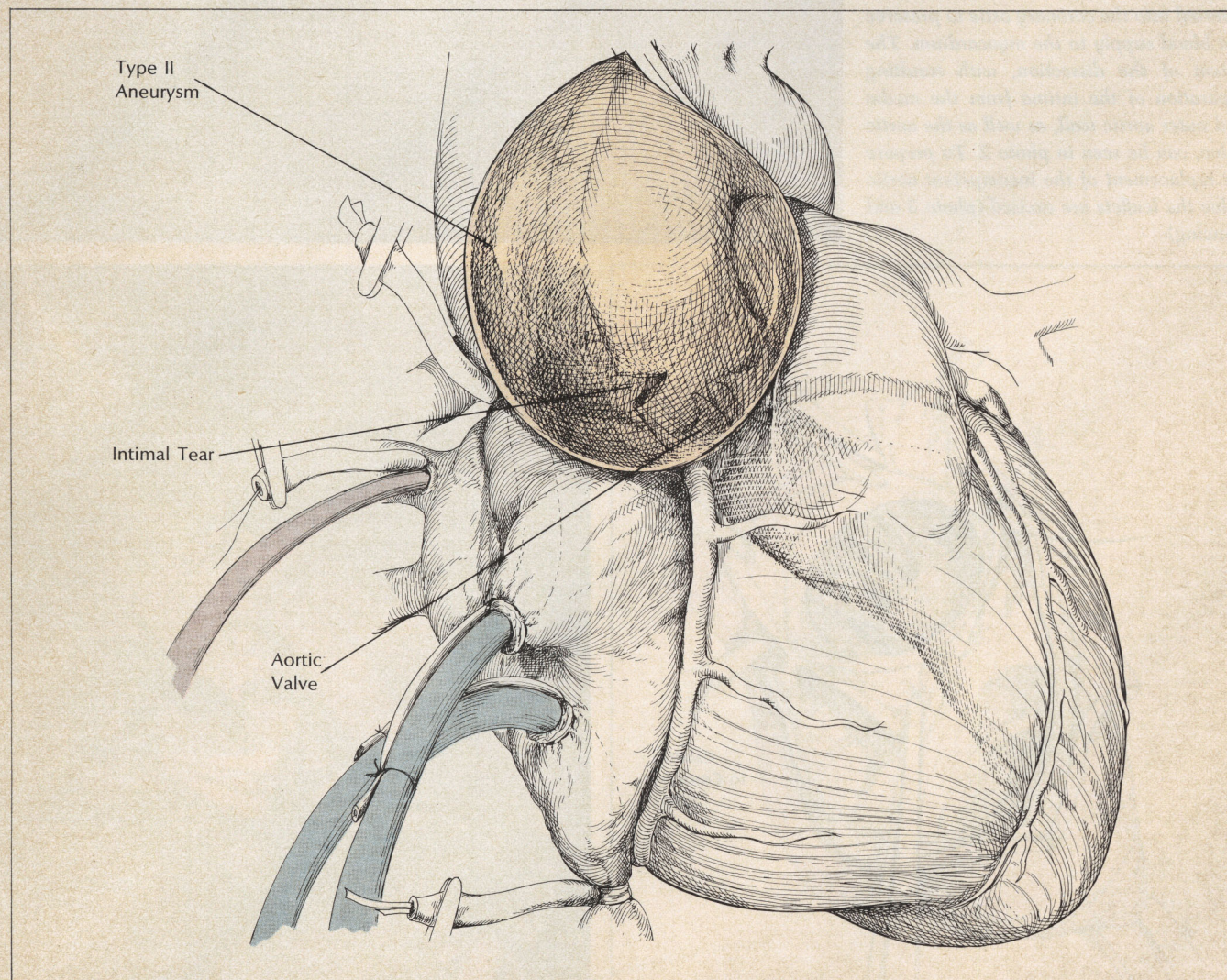

In the initial stages of surgery for type II dissecting aneurysm, the vena cavae are cannulated via the right atrium, the femoral artery then cannulated, a sump placed in the left atrium via the pulmonary vein, and cardiopulmonary bypass started. The aneurysm is then cross-clamped just proximal to the origin of the innominate artery, preparatory to decompression.
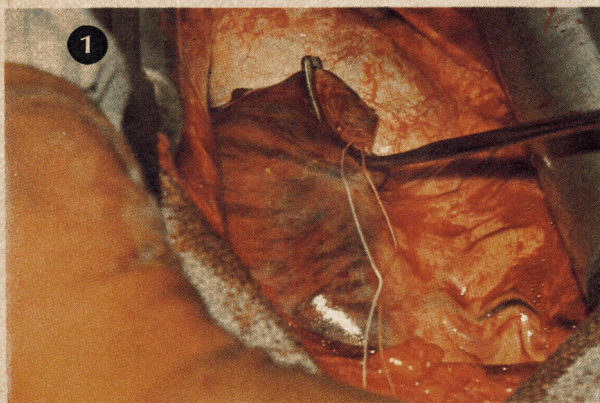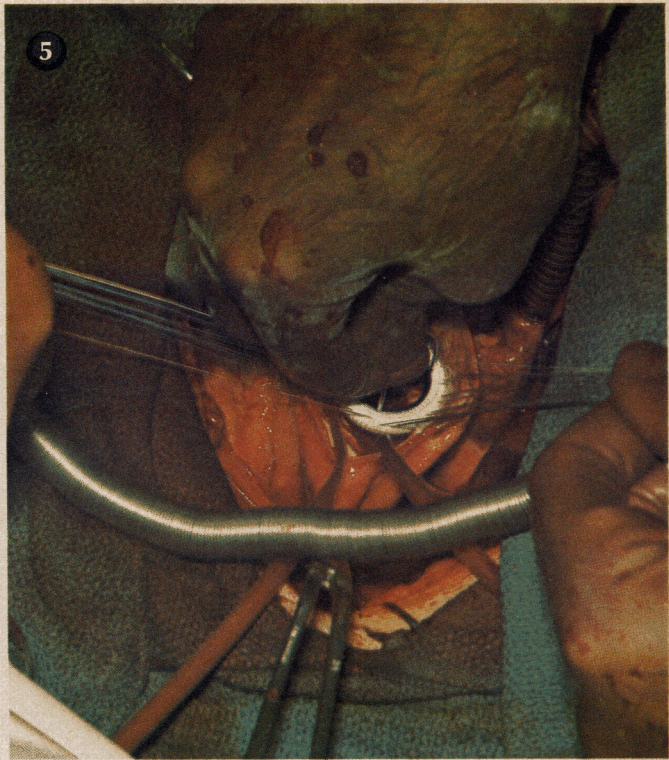
The aneurysm has been approached through a median sternotomy and is clearly visible (1) as a ballooned-out, whitish area. Cardiopulmonary bypass is instituted and, after the ascending aorta has been clamped just proximal to the origin of the innominate vessels, the aneurysm is entered and decompressed. Catheters are inserted into the coronary ostia to preserve the blood supply to the myocardium. The extent of the dissection, with resulting separation of the intima from the media and outer aortic wall, as well as the aortic valve, can be seen in photo 2. To prepare for replacement of the incompetent aortic valve the leaflets are excised (photo 3 and drawing).


Valve Leaflet

Cannula to Coronary Artery
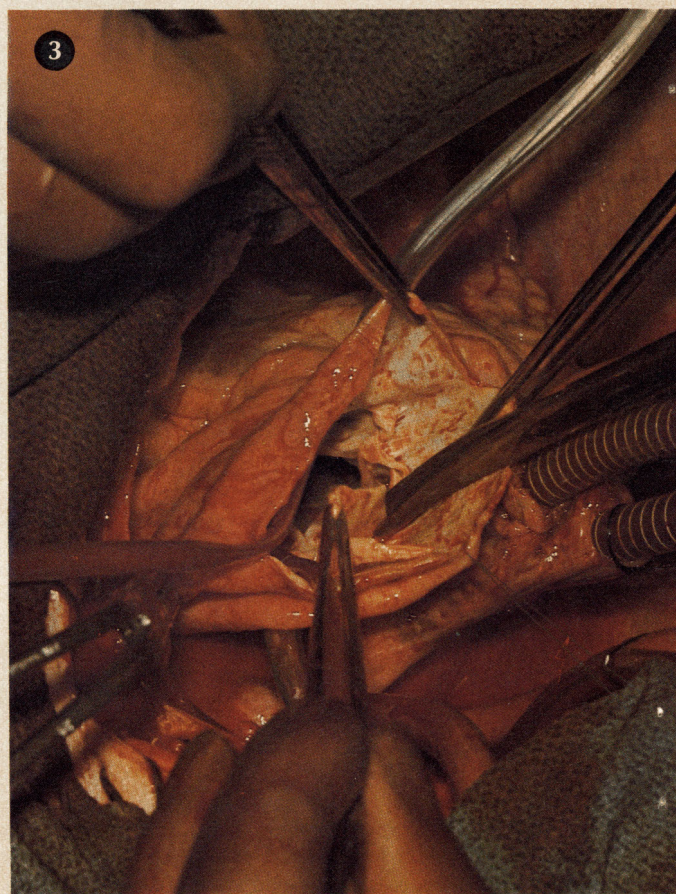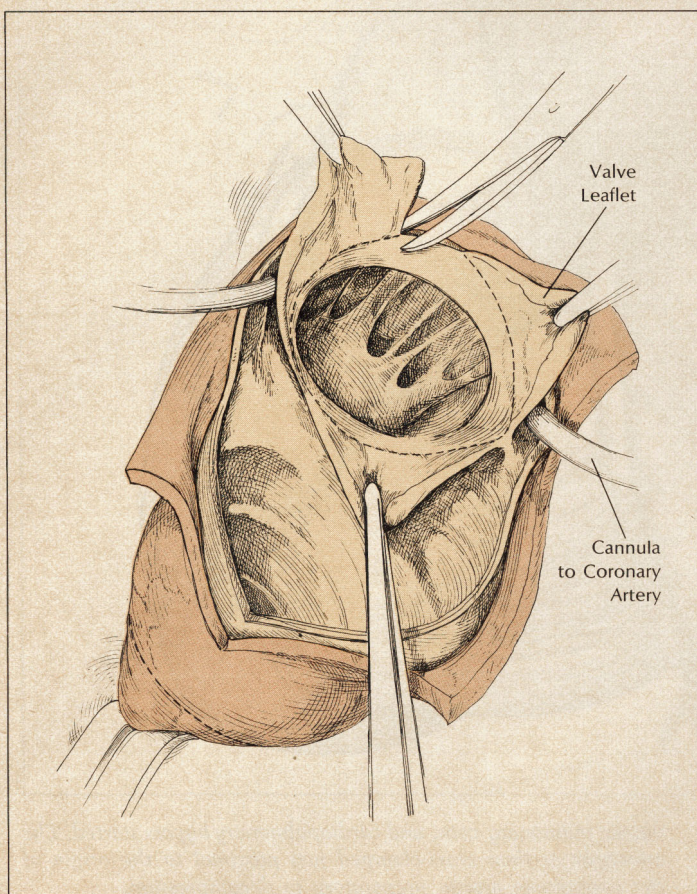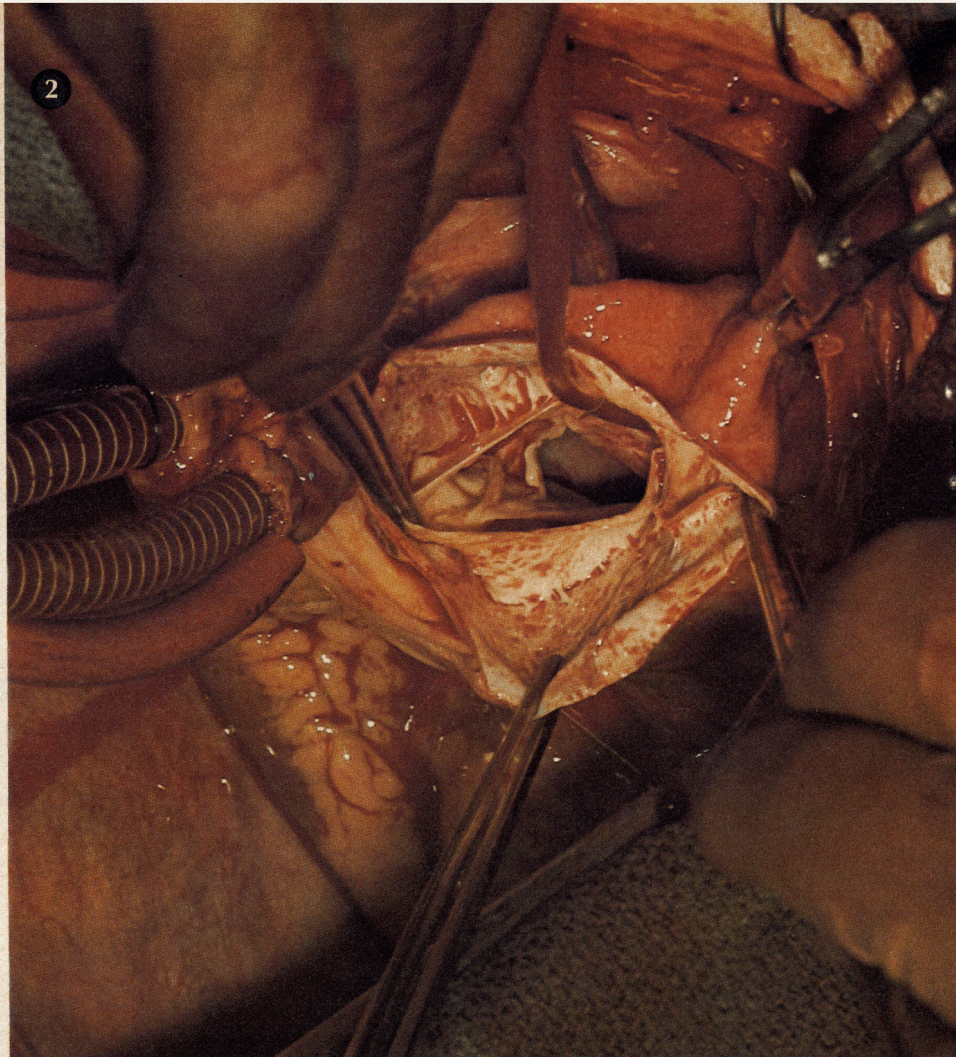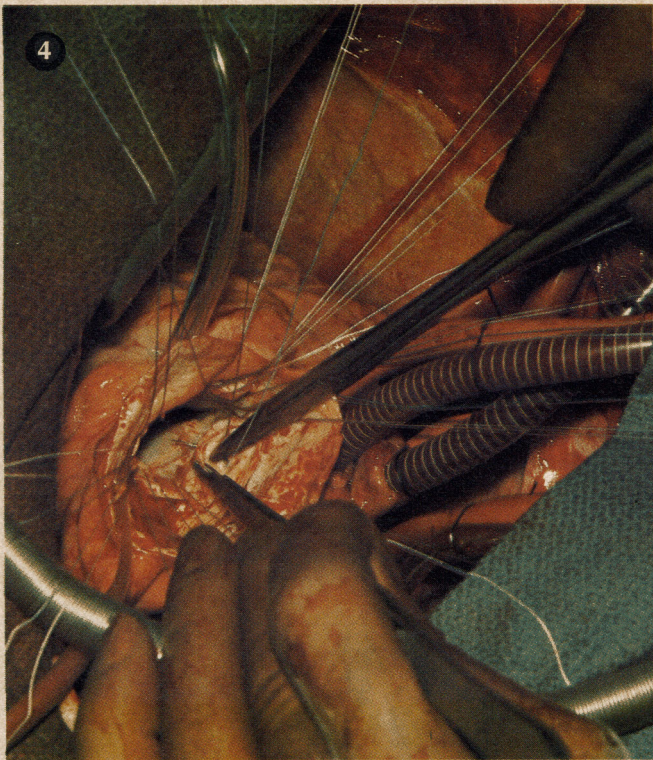
After the leaflets of the aortic valve have been excised, anchoring sutures for the pyrolytic DeBakey caged-ball prothesis are placed around the entire circumference of the aortic anulus (4). The cuff of the valve cage is then inserted deep into the anulus (5) and the sutures tied. The ball itself is to be inserted at a later stage in the operation. Next the dissection of the aneurysm is continued distally towards the innominate vessels and the intima and media are carefully stripped from the posterior wall of the aneurysm (6). As is usual in this type of aortic aneurysm, the outer layer is found to be quite thin.
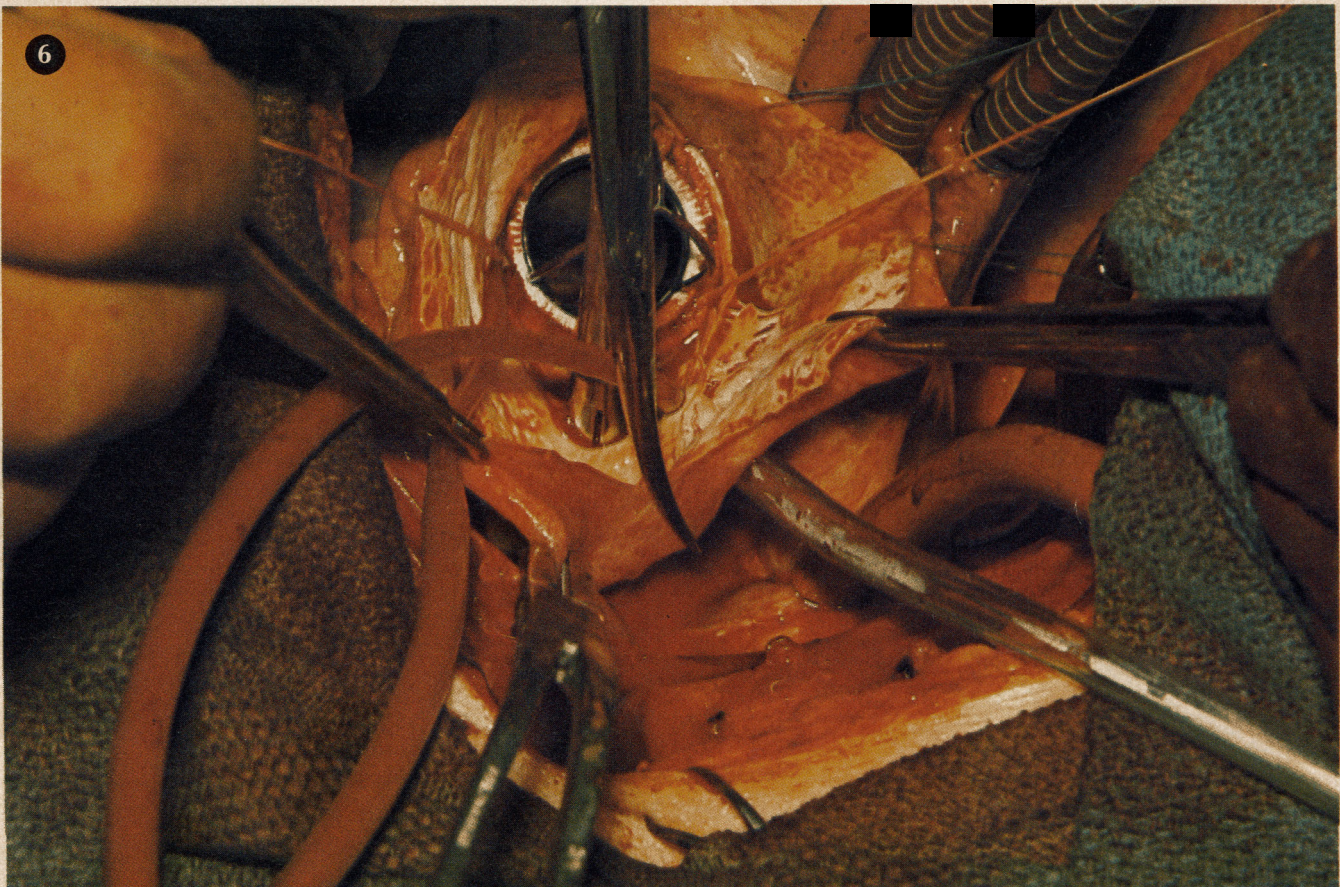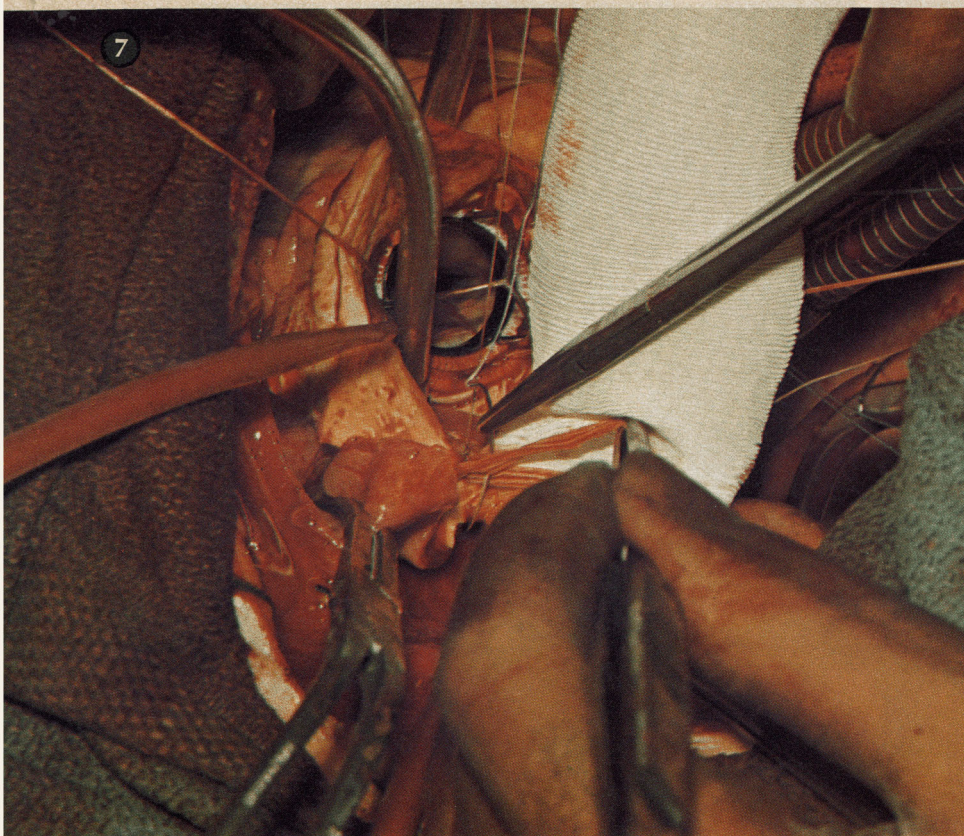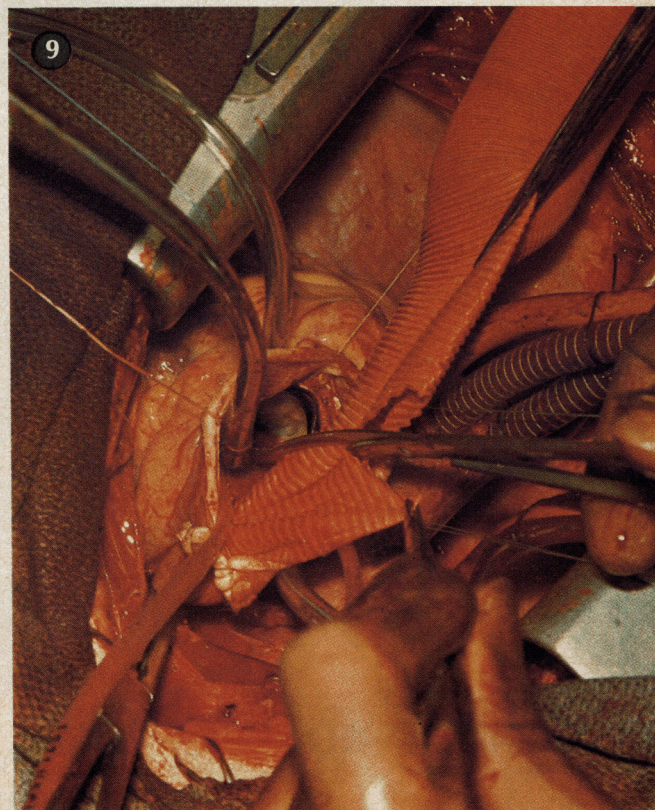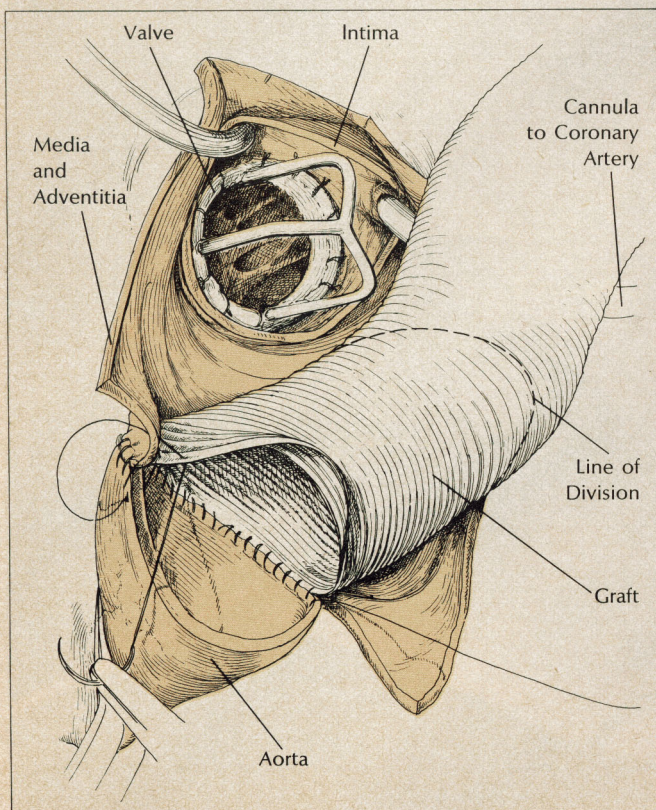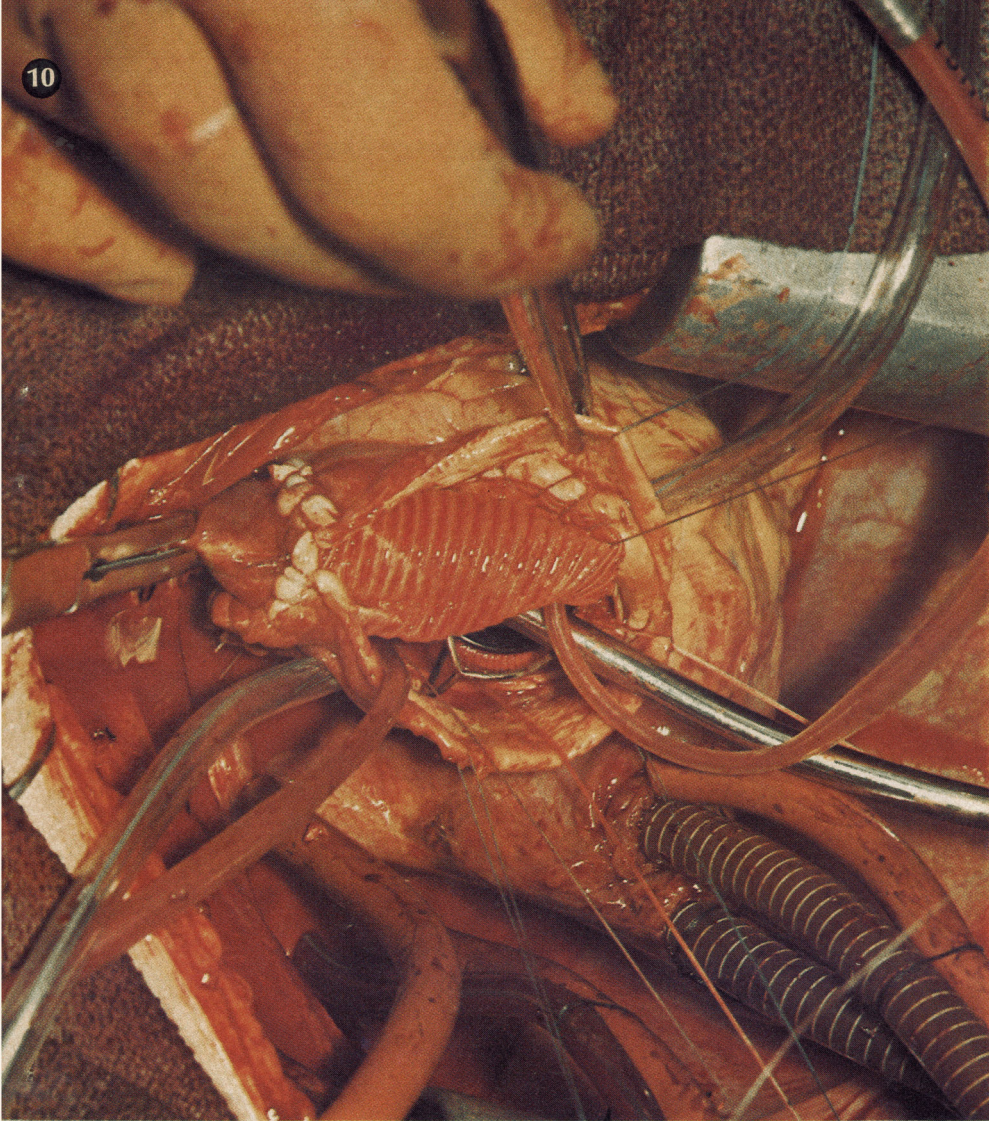
A length of 35 mm crimped, woven Dacron tubing is next anastomosed to the distal portion of the ascending aorta (7 and 8), using a continuous suture. The sutures are put through the outer wall of the aneurysm, which remains attached to the aorta distally and to the aortic root proximally. After the prosthesis has been secured distally, it is bias-cut by sight to fit the curved area of the aortic root (9 and drawing at left below) and anchored posteriorly as end-to-end anastomosis is begun. The outer wall of the aneurysm, from which the media and intima were removed earlier, will be closed over the prosthesis.


Valve · Intima · Cannula to Coronary Artery · Media and Adventitia · Line of Division · Graft · Aorta

The suture line is continued anteriorly (10), with the anastomosis now almost complete. Before the final sutures are put in place, the ball of the valve, which was left out to lessen the risk of entrapment of air, is inserted into the cage (drawing at right below). The catheters to the coronary arteries are to be withdrawn next and the anastomosis completed, with the externa, seen being held apart with traction sutures in photo 11, to be approximated over the anterior portion of the prosthesis. In suturing the prosthesis in place, care is taken to put the stitches through the intima left intact as well as the media and adventitia at both proximal and distal ends. This step is important to prevent recurrence of the aneurysm or otherwise compromise the results of surgery.
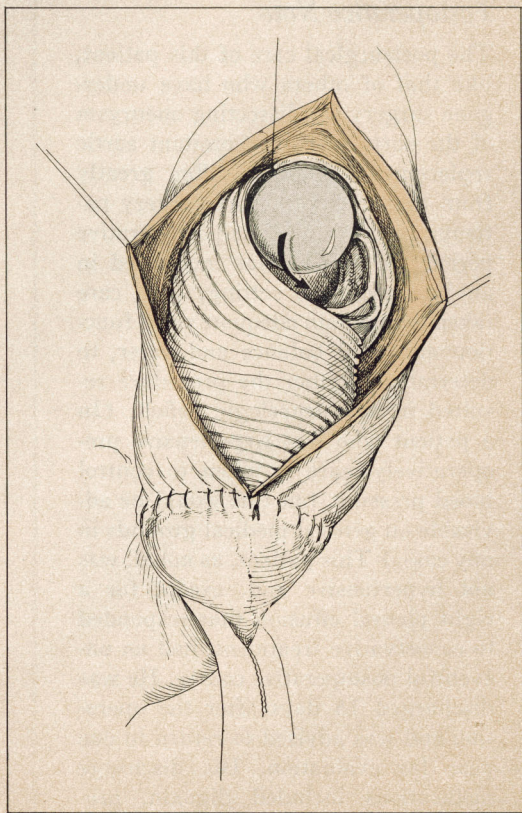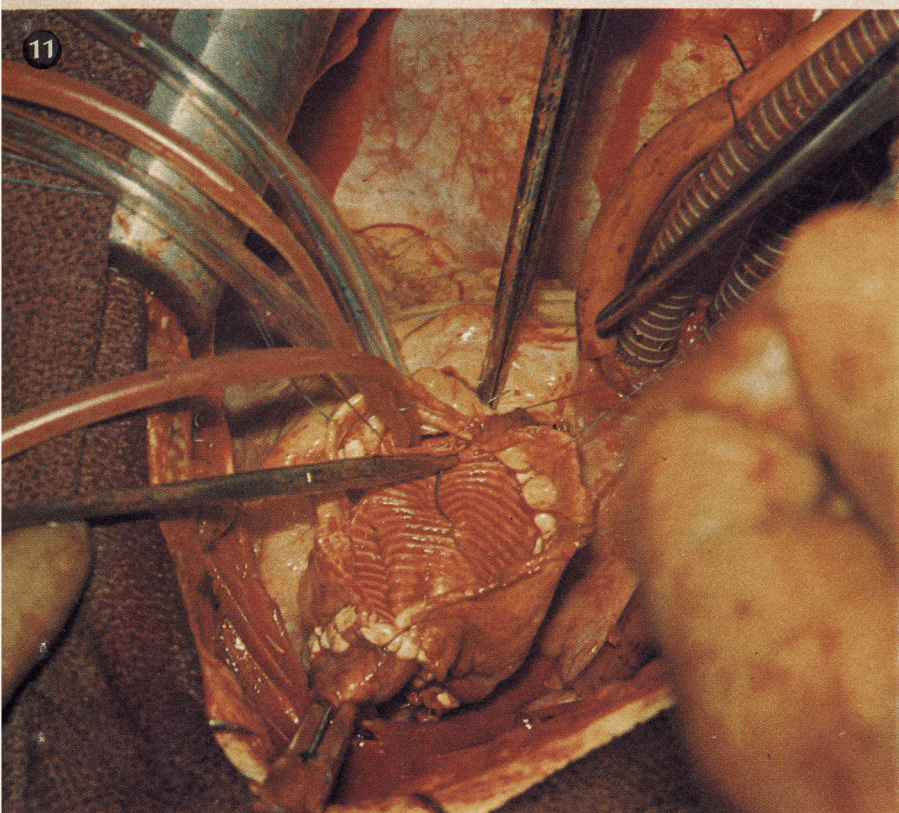
In the final stages of the procedure, just before the proximal anastomosis of the graft is completed, suction on the sump in the left atrium is discontinued to allow blood to fill the left ventricle and displace the air, the coronary catheters are removed, and the anastomosis is completed. The patient is placed in the head-down position, and a needle is inserted into the ascending aorta just proximal to the anastomosis to permit any remaining air in the heart to be removed. The proximal aortic occlusion clamp is released, the sump catheter in the left atrium is removed, and the opening is closed by tying the purse-string suture. Cardiopulmonary bypass is then discontinued and blood allowed to flow through the ascending aorta once more. The last sutures are then placed in the externa (12) to complete its closure over the graft. Covering the graft in this fashion leads to the rapid regrowth of the adventitia over the prosthesis and helps ensure the success of the procedure. As can be seen in the drawing, the Dacron prosthesis now replaces the entire ascending aorta while the DeBakey caged-ball valve governs the heart's outflow.
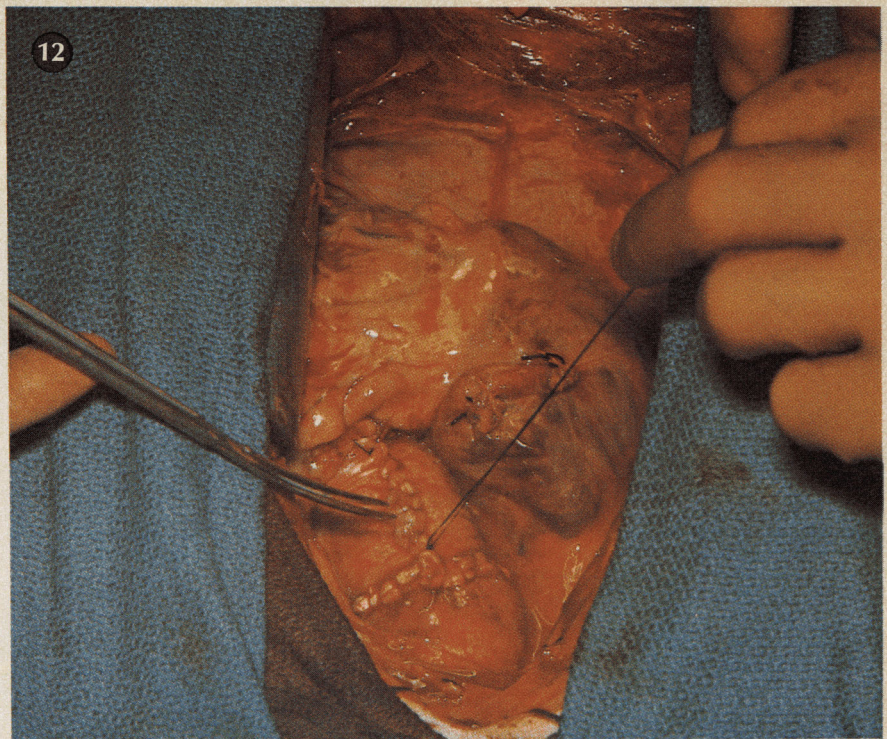
Postoperative Note

The postsurgical care of this patient, like that of others who have undergone surgery for dissecting aneurysm of the aorta, with concomitant aortic replacement, did not differ greatly from that of other heart surgery patients. In the immediate postoperative period the patient was monitored in an intensive care setting, with care taken to maintain adequate renal function and fluid balance and, later, to determine whether the procedure resulted in any neurologic deficit. (In a patient who was hypertensive preoperatively, care is taken to control blood pressure, but this would be advisable on general medical grounds in any case.) This patient, as noted, was not hypertensive and, except for a right pleural effusion that responded to conservative treatment, had an uneventful postoperative course. He was discharged 15 days after the operation and was advised to return in one year for evaluation. His discharge medications included digoxin, procainamide, and an iron supplement.



Graft

Closure of
Adventitia

Valve
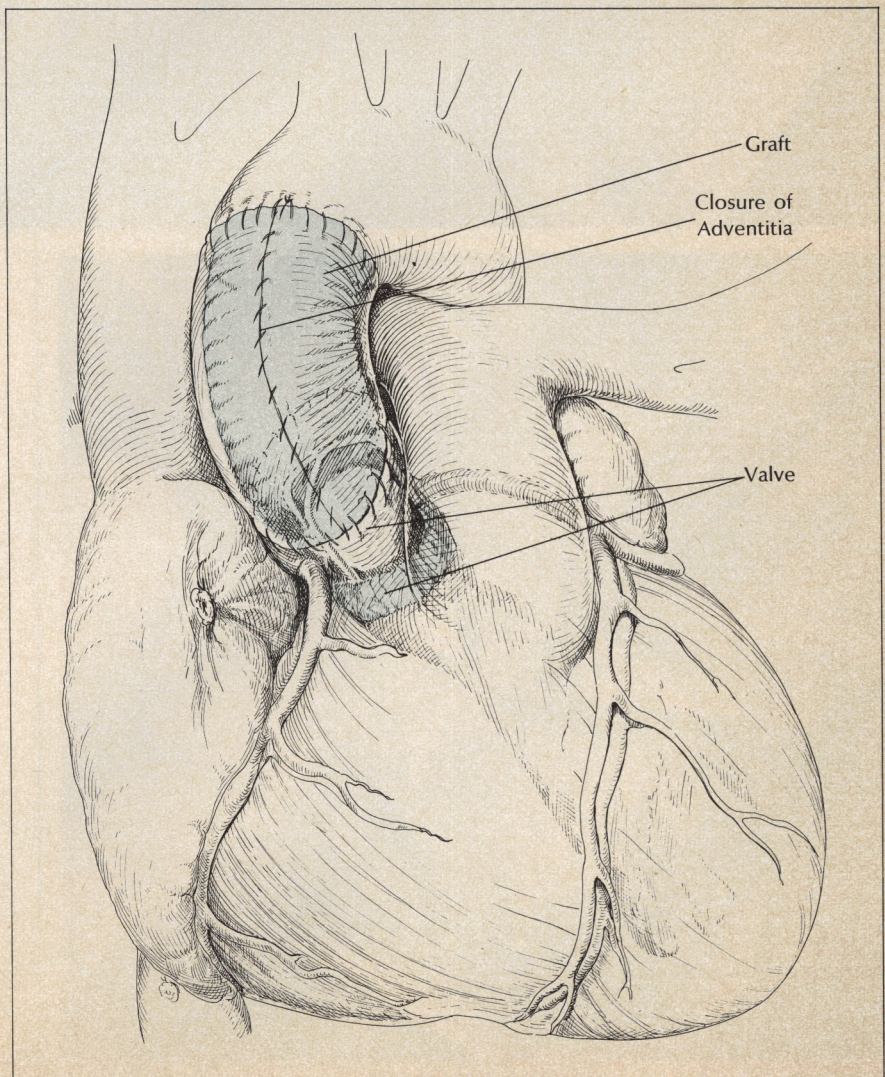
The Case in Context

Dissecting aneurysm of the aorta, of whatever type, has long been dreaded for its catastrophic potential. Until surgical means of correction were perfected during the past 15 years, the outcome in three fourths of all cases was death. Up to then patients hospitalized because of an acute episode could be offered only palliative measures and in essence were considered terminal.

Those who unpredictably survived did so because of spontaneous healing of the aneurysm, a phenomenon that has been recognized for more than a century. In most of these cases the aneurysm would rupture into the lumen of the aorta at or near its distal end, creating a two-channeled but functioning aorta. The earliest attempts at surgical correction tried to duplicate this natural phenomenon. But it became apparent that such surgery was at best palliative, for there was no way to strengthen the adventitia or to prevent resumption of the dissecting process and/or rupture of the thinned aortic wall.

After a few trials with this technique we concluded that some type of excisional therapy, with replacement or bypass of the diseased aortic segment, employing either a homograft or a prosthesis, offered the best prospects for surgical correction. In the early years of such surgical correction, however, the advantages it offered over the previous therapeutic nihilism were none too clear. Operative mortality during the first three years we were following this approach was 41%. It has since dropped steadily as our understanding of the various types of aneurysms and the best way to handle them has improved, so that it now stands at about 5%. This means that some 95% of all patients undergoing surgery to correct dissecting aneurysm, with aortic valve replacement when indicated, can return to a
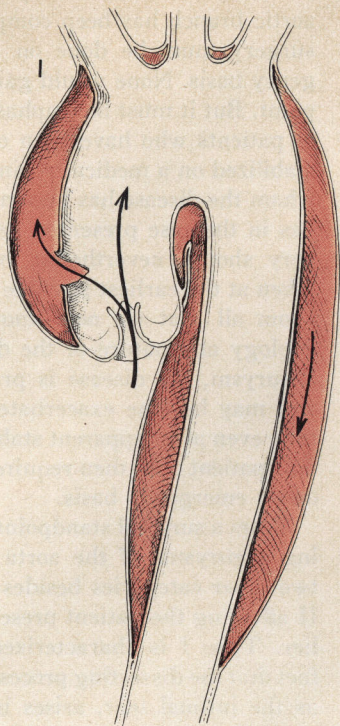
On the basis of the surgical therapy required, the major types of dissecting aneurysms of the aorta fall into three main categories, shown here schematically. In type I the intimal tear initiating the dissecting process occurs in the ascending aorta and the dissection then continues for a variable distance, but usually involves the entire aorta, including its major terminal branches. Ordinarily, however, the dissection does not involve the entire circumference of the vessel. Commonly (but not invariably) the commisural support of the aortic valve is weakened, causing regurgitation.

Type II dissecting aneurysms likewise begin with an intimal tear in the ascending aorta, but the dissecting process is limited to the ascending aorta itself so that the relatively thin outer aortic layer often gives the appearance of a fusiform aneurysm. The aortic valve usually is incompetent owing to loss of commisural support or dilatation of the anulus.
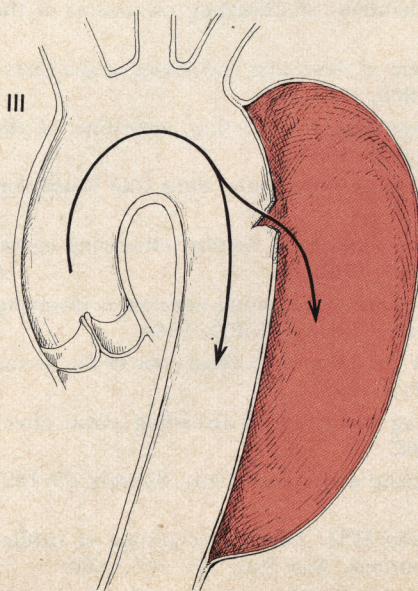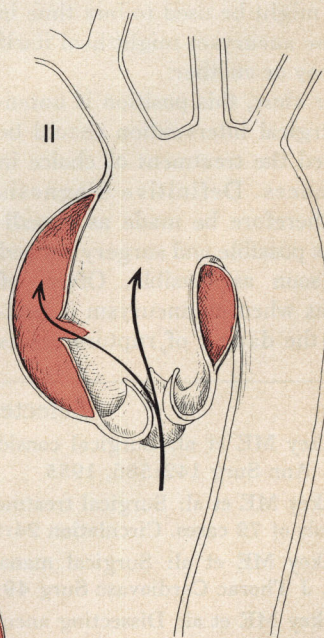
In the type III dissecting aneurysm the intimal tear occurs in the proximal descending thoracic aorta, usually at or just distal to the origin of the left subclavian artery in the region of the attachment of the ligamentum arteriosus. The dissecting process extends distally a varying distance, sometimes going beyond the bifurcation and into the terminal branches.

normal, productive life, free from the fear of an impending catastrophe and unhampered by the symptoms of aortic insufficiency.

During the years that our surgical approach was being improved, the introduction of a number of drugs – basically, antihypertensives and inotropic agents – made it possible to influence the factors bearing on the immediate outcome in dissecting aneurysm. This was particularly true in patients undergoing an acute episode and seen within a week or two of onset. Such drugs have made it possible to tide over a great many patients who might otherwise have died, allowing their condition to stabilize so that definitive surgery could be done under more favorable circumstances. This helpful development has led some physicians to suggest that medical management alone might suffice in many patients with dissecting aneurysm. The argument is that chemotherapy might be used to buy time in which the aneurysm might heal itself in one way or another.

In our view, this position is untenable. Surgical intervention should be considered the treatment of choice in all instances. Definitive diagnosis should therefore be made as expeditiously as possible and surgery carried out as soon as feasible. Often, of course, as when an aneurysm is leaking and the danger of rupture seems imminent, or there is overwhelming aortic valvular insufficiency or a major aortic branch has been compromised, surgery must be done on an emergency basis. None would gainsay this point. But it must be emphasized that in patients who have been ostensibly stabilized on a medical regimen, or in whom the disease has become chronic (as in the case presented here), surgery should nevertheless be undertaken at the earliest possible moment. From all that we now know of the etiology and nature of the dissecting aneurysm, the disease is progressive and may become exacerbated at any time even after apparent stabilization. The patient may then require surgery on an emergency basis.

From a surgical standpoint, dissecting aneurysms of the aorta fall into two other categories besides the type II affecting the patient presented earlier. Type I is characterized by the fact that the dissecting process, as well as the intimal tear, arises in the ascending aorta and extends distally beyond the origin of the innominate vessels for a variable distance, sometimes into the major terminal branches. The dissection may involve the entire circumference of the aorta but is usually incomplete. Aortic valve insufficiency is a frequent but not invariable concomitant. Type III is characterized by the fact that the dissecting process commences in the descending aorta, usually at or just distal to the origin of the left subclavian artery and extends distally for a varying distance (see drawings, page 85).

The particulars of surgery, obviously, differ for each type of aneurysm. But regardless of the nature or extent of the aneurysm, the fact remains that excisional therapy and prosthetic repair provide the only definitive treatment. This point was clearly established a few years ago when a study of a series of 425 patients treated by nonsurgical means showed that only 7% were alive one year after diagnosis, as compared with 70% of our own cases at the end of a like period. Even more impressive was the five-year survival rate: 50% of our surgically treated patients were alive, as contrasted with fewer than 1% of those treated by nonsurgical means.

Diagnosis is not always as straightforward as in the patient described. Not all cases begin with a single, definable episode of chest pain; instead there may be recurrent pain in the back or epigastric area and in these cases there may be widening of the mediastinal shadow on x-ray. Dissection affecting the ascending aorta may sometimes present as cardiac tamponade – a medical and surgical emergency – or with the symptoms of aortic insufficiency and presumed heart failure or myocardial infarction.

Quite frequently also the patient may be pallid and appear to be in shock. But almost invariably the blood pressure will be inappropriately high for shock and the patient may in fact be hypertensive, although less so than before onset of symptoms. In those patients who are relatively hypertensive, of course, it is essential to reduce the blood pressure so as to minimize the risk of aneurysmal rupture.

The surest means of establishing the diagnosis is cardiac catheterization. This should be done early and, if at all feasible, by the retrograde approach so as to obtain the best pictures possible. In most instances, the true lumen of the aorta and the false lumen of the aneurysm can be clearly visualized so that there can be no doubt concerning the diagnosis. Once the diagnosis has been made – to repeat – surgery should be undertaken as soon as possible. Any other approach courts disaster. □

Selected Reading

DeBakey ME et al: Surgical considerations of dissecting aneurysms of the aorta. Ann Surg 142:586, 1955

DeBakey ME et al: Surgical treatment of dissecting aneurysms of the aorta: analysis of 72 cases. Circulation 24:290, 1961

DeBakey ME et al: Surgical management of dissecting aneurysms of the aorta. J Thorac Cardiovasc Surg 49:130, 1955

DeBakey ME et al: Dissecting aneurysms of the aorta. Surg Clin North Am 46:1045, 1966

DeBakey ME et al: Aneurysms of the aortic arch: factors influencing operative risk. Surg Clin North Am 42:1543, 1962

DeBakey ME et al: Surgical management of dissecting aneurysms involving the ascending aorta. J Cardiovasc Surg (Torino) 5:200, 1964

Hirst AE Jr, Johns VJ Jr, Kime SW Jr: Dissecting aneurysms of the aorta: a review of 505 cases. Medicine 37:217, 1958

Hufnagel CA, Conrad PW: Dissecting aneurysms of ascending aorta: direct approach repair. Surgery 51:84, 1962

Hume DM, Porter RR: Acute dissecting aortic aneurysms. Surgery 53:122, 1963

Muller WH Jr, Damman FJ, Warren WD: Surgical correction of cardiovascular deformities in Marfan's syndrome. Ann Surg 152:506, 1960

Shennan T: Dissecting aneurysms (Med Res Council, Spec Rep Series No 193), London, HMSO, 1934